In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: July 27, 2020

ANNA CONTE,	*	No. 17-403V
Petitioner,	*	Special Master Sanders
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Ruling on the Record; Insufficient Proof; Influenza ("flu") Vaccine; Guillain-Barré syndrome ("GBS")
Respondent.	*	

Isaiah R. Kalinowski, Maglio Christopher and Toale, PA, Washington, DC, for Petitioner.
Darryl R. Wishard, United States Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On March 22, 2017, Anna Conte ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("Vaccine Program" or "the Program").[2] Pet. at 1, ECF No. 1. Petitioner alleges that the influenza ("flu") vaccine she received on September 30, 2015, caused her to develop Guillain-Barré syndrome ("GBS"). *Id.* The information in the record, however, does not show an entitlement to an award in the Program.

I. Procedural History

Petitioner filed her petition on March 22, 2017. *Id.* On the same day, Petitioner filed twelve exhibits consisting of medical records. *See* Pet'r's Exs. 1–13,[3] ECF Nos. 5-2–5-10, 6-2–6-6. Petitioner filed an additional medical records exhibit and a statement of completion on April 21, 2017. *See* Pet'r's Ex. 14, ECF No. 11-2; ECF No. 12.

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[1] This decision shall be posted on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), a party has 14 days to identify and move to delete medical or other information that satisfies the criteria in § 300aa-12(d)(4)(B). Further, consistent with the rule requirement, a motion for redaction must include a proposed redacted decision. If, upon review, I agree that the identified material fits within the requirements of that provision, such material will be deleted from public access.

[2] The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-10 to -34.

[3] Petitioner filed exhibit 7 in two parts. *See* Pet'r's Exs. 7.1–7.2.

On August 21, 2017, Respondent filed a status report in which he indicated that he had "completed his preliminary review of the records and has not identified any additional records that are missing at this time." ECF No. 18. The next day, I ordered Respondent to file his Rule 4(c) report by October 20, 2017. Non-PDF Order, docketed Aug. 22, 2017. Respondent filed a motion for extension of time on October 22, 2017, ECF No. 19, which I granted, *see* Non-PDF Order, docketed Oct. 23, 2017, and Respondent filed his Rule 4(c) report on November 2, 2017, Resp't's Report, ECF No. 20. Respondent argued in his report that "Petitioner is not entitled to compensation under the terms of the Vaccine Act[]" and requested that I dismiss the petition. *Id.* at 12.

I held a Rule 5 conference with the parties on November 15, 2017, *see* Min. Entry, docketed Nov. 15, 2017, and ordered Petitioner to file an expert report by January 31, 2018, ECF No. 21. Petitioner filed three affidavits on December 19, 2017. Pet'r's Exs. 16–18, ECF Nos. 24-2–24-4. On January 24, 2018, Petitioner filed an expert report authored by Zurab Nadareishvili, M.D., along with his curriculum vitae and sixteen pieces of supporting medical literature. Pet'r's Exs. 19–36, ECF Nos. 25-2–25-3, 26-2–26-10, 27-2–27-8. The next day, I ordered Respondent to file a responsive expert report by March 26, 2018. Non-PDF Order, docketed Jan. 25, 2018.

Petitioner filed an additional exhibit consisting of medical records on March 13, 2018. Pet'r's Ex. 37, ECF No. 28-2. On March 23, 2018, Respondent filed a motion for extension of time to submit a responsive expert report, ECF No. 29, which I granted, ECF No. 30. Respondent subsequently filed an expert report authored by Vinay Chaudry, M.D., along with his curriculum vitae and one piece of supporting medical literature on May 25, 2018. Resp't's Exs. A–C, ECF Nos. 31-1–31-3. On May 30, 2018, I ordered Petitioner to file a responsive expert report by July 24, 2018. Non-PDF Order, docketed May 30, 2018. On July 23, 2018, Petitioner filed a responsive supplemental expert report by Dr. Nadareishvili and one piece of supporting medical literature. Pet'r's Exs. 38–39, ECF Nos. 32-2–32-3.

On September 19, 2018, I conducted a review of Petitioner's medical records and both parties' expert reports filed to that point and issued an order containing a list of questions for each expert to address by November 19, 2018. ECF No. 33. Petitioner filed Dr. Nadareishvili's responses to my questions, along with two pieces of medical literature, on November 16, 2018. Pet'r's Exs. 40–42, ECF Nos. 34-1–34-3.

On the same day, Petitioner filed a motion to exclude the testimony of Respondent's expert, Dr. Chaudry, because "it does not meet the legal threshold of *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), in that it does not apply scientific methodology, and does not provide evidence to support its claim." Pet'r's Mot. to Exclude Testimony of Dr. Chaudry at 2, ECF No. 35. On November 19, 2018, Respondent filed a motion for extension of time to respond to my questions and a request that I stay Petitioner's pending motion because "it would be premature to decide [P]etitioner's motion" while "Dr. Chaudhry is still in the process of providing written testimony for the Court's consideration[,]" ECF No. 36. I issued an order granting Respondent's motion on November 20, 2018, and ordered Respondent to file his expert's responses to my questions by January 18, 2019. ECF No. 37. In the same order, I also stayed Petitioner's motion to exclude Dr. Chaudry's testimony. *Id.*

Petitioner filed an additional affidavit on December 22, 2018. Pet'r's Ex. 43, ECF No. 38-2. On February 28, 2019, Respondent filed an expert report containing Dr. Chaudry's responses to my questions and to Dr. Nadareishvili's most recent expert report, along with seven pieces of supporting medical literature. Resp't's Exs. D–K, ECF Nos. 39-1–39-8. Petitioner filed a status report on March 12, 2019, in which she indicated that she did not believe that any "further expert reports are necessary." ECF No. 40.

After conducting a review of both parties' responses to my previous questions and the accompanying medical literature, I issued an order on April 11, 2019, in which I directed additional questions to the parties' experts and ordered the parties to file their responses by May 13, 2019. ECF No. 42. Petitioner filed Dr. Nadareishvili's responses and three pieces of supporting medical literature on April 22, 2019. Pet'r's Exs. 44–47, ECF Nos. 43-1–43-4. After one extension of time, Respondent filed Dr. Chaudry's responses to my questions and three pieces of supporting medical literature on May 20, 2019. *See* ECF Nos. 44–45; Resp't's Exs. L–O, ECF No. 46-1–46-4. On June 24, 2019, Petitioner filed a status report in which she again indicated that she did not believe that any "further expert reports are necessary." ECF No. 47.

I held a status conference with the parties on July 31, 2019, Min. Entry, docketed July 31, 2019, during which "[t]he parties discussed how the case should proceed and determined that it should be decided on the record[,]" ECF No. 48. I ordered Petitioner to file any additional evidence that she would like to be considered by August 30, 2019. *Id.* On August 30, 2019, Petitioner filed one piece of medical literature. Pet'r's Ex. 48, ECF No. 49-2.

Neither party has filed any additional evidence. This matter is now ripe for consideration.

II. Factual Background

A. Relevant Medical History

On July 23, 2015, Petitioner presented to S. Brett Whitfield, M.D., an orthopedic surgeon, for a consultation regarding "severe arthritis" and a "problem with her right finger." Pet'r's Ex. 9 at 12. Dr. Whitfield noted that Petitioner had "had injections" for her severe arthritis in the past and had recently received an injection in one of her knees. *Id.* Petitioner reported doing "very well with the [recent] injection." *Id.* In addition to her knee arthritis, Petitioner complained that, a day prior, "[s]he was lifting something and felt some pain[]" in her right hand. *Id.* Dr. Whitfield's assessment included "[m]allet finger,"[4] "osteopenia[5] . . . [of her] bilateral lower extremities[,]" and "[s]evere patellofemoral arthritis[in her] bilateral knees." *Id.* at 15. Dr.

[4] A mallet finger is defined as "partial permanent flexion of the terminal phalanx of a finger caused by a ball or other object striking the end or back of the finger, resulting in rupture of the attachment of the extensor tendon." *Dorland's Illustrated Medical Dictionary* 701 (33rd ed. 2020) [hereinafter "*Dorland's*"].

[5] Osteopenia is defined as "1. any decrease in bone mass below the normal[; or] 2. reduced bone mass due to decrease in the rate of osteogenesis to the extent that there is insufficient compensation for normal bone lysis." *Dorland's* at 1329. Lysis is defined as "dissolution or destruction of an organ or structure, such as the destruction of bone by loss of calcium . . . ." *Id.* at 1075.

Whitfield placed Petitioner's injured finger in a "stack splint" and prescribed her Mobic[6] for her knees, although he noted that she did not need any additional injections at that time. *Id.*

On September 30, 2015, Petitioner presented to her primary care physician ("PCP") with chief complaints of "[b]ack pain, hypertension, and dyslipidemia."[7] Pet'r's Ex. 10 at 42. The history of present illness ("HPI") section of the note reflects that Petitioner had "a long history of pain across [her] lower back[, which was a] little bit more frequent lately." *Id.* Upon exam, Petitioner's blood pressure was elevated. *Id.* Her PCP prescribed her Tylenol for her back pain and ordered an x-ray of her lumbosacral spine. *Id.* at 43. Petitioner also received the flu vaccine at issue at this visit. *Id.* at 323; Pet'r's Ex. 1 at 1. Petitioner underwent a lumbosacral spine x-ray on October 1, 2015, which revealed "known degenerative disc disease at L4-5 and L5-S1 with anterior spondylolisthesis[8] of L4 relative to L5." Pet'r's Ex. 10 at 239. It also showed "[t]iny calcification in the projection of the right upper quadrant which measures approximately [nine millimeters]" and "[s]cattered arterial atherosclerotic plaque formation of aorta." *Id.*

Petitioner returned to her PCP on October 28, 2015, with complaints of "[n]umbness [in] both hands, pain at the base of both thumbs[, and h]ypertension." *Id.* at 40. Petitioner reported that "[f]or the past [two] or [three] weeks[, she] has had numbness in both hands[, e]specially the fingertips." *Id.* She explained that she had to "wake[] up through the night and . . . shake her hands to get the feeling back." *Id.* She noted that "[a]bout [two] weeks ago[, she] had pain[at the] base of the right thumb[,]" and "now [has] pain at the base of the left thumb." *Id.* Her PCP ordered nerve conduction studies ("NCS") of both of Petitioner's hands and prescribed tramadol[9] for her osteoarthritic pain. *Id.* at 41. Two days later, on October 30, 2015, Petitioner called her PCP and reported that "she may have to go to the [emergency room] because of the pain in her hands" and "that the med[ication] [the PCP had] prescribed doesn't help" her pain. *Id.* at 39.

On November 2, 2015, Petitioner presented to Beckley Appalachian Regional Healthcare ("BARH") hospital with complaints of "back pain radiating down [her] right arm [for one] day." Pet'r's Ex. 4 at 179–81. Petitioner reported that she also experienced "some nausea and chest tightness[]" and that "Tylenol helps some with [the] pain." *Id.* at 181. She explained "that she has had back pain off and on for several years secondary to osteoarthritis[10] and degenerative joint

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[6] Mobic is the "trademark for a preparation of meloxicam." *Dorland's* at 1154. Meloxicam is "a nonsteroidal anti-inflammatory drug used in the treatment of osteoarthritis; administered orally." *Id.* at 1111.

[7] Dyslipidemia is defined as "an abnormality in, or an abnormal amount of, lipids and lipoproteins in the blood . . . ." *Dorland's* at 572.

[8] Spondylolisthesis is "the forward displacement . . . of one vertebrae over another, usually of the fifth lumbar over the body of the sacrum, or of the fourth lumbar over the fifth, usually due to a developmental defect in the pars interarticularis." *Dorland's* at 1725. The sacrum is "the triangular bone just below the lumbar vertebrae[.]" *Id.* at 1635.

[9] Tramadol hydrochloride is "an opioid analgesic used for the treatment of moderate to moderately severe pain following surgical procedures and oral surgery; administered orally." *Dorland's* at 1920.

[10] Osteoarthritis is defined as "a non-inflammatory degenerative joint disease seen mainly in older persons, characterized by degeneration of the articular cartilage, hypertrophy of bone at the margins, and changes in the synovial membrane. It is accompanied by pain, usually after prolonged activity, and stiffness, particularly in the morning or with inactivity." *Dorland's* at 1326. Osteoarthritis is also known as degenerative joint disease. *Id.*

disease."[11] *Id.* at 97. Petitioner's blood pressure was elevated upon exam. *Id.* While in the emergency room, Petitioner "went to the bathroom and had [a] syncopal episode[12] while there[,]" after which Petitioner "denie[d] pain but" complained of "severe weakness." *Id.* at 182. Petitioner was admitted for further evaluation. *Id.*

While admitted, Petitioner underwent computed tomography[13] ("CT") scans of her chest, abdomen, and pelvis, which were all unremarkable. *See* Pet'r's Ex. 9 at 43–47. Doctors also ruled out acute cardiopulmonary disease and acute coronary syndrome. Pet'r's Ex. 4 at 98. Petitioner also had a normal neurological exam. *Id.* at 98, 181. Petitioner was assessed with atypical chest pain that doctors thought was "most likely secondary to uncontrolled hypertension." *Id.* at 98. Doctors noted that they had brought Petitioner's blood pressure "under control by adding lisinopril[14] to her regimen, as well as [administering] IV hydralazine."[15] *Id.* The discharge note reflects that Petitioner's "chest discomfort did not return during her stay[]" in the hospital, and that upon discharge, her blood pressure "was well controlled . . . ." *Id.* at 98–99. Doctors directed Petitioner to follow-up with her PCP. *Id.* at 99.

On November 9, 2015, Petitioner walked in to her PCP's office, stated that she was still experiencing pain, and requested "an xray [sic.] to see whats [sic.] going on with her hands." Pet'r's Ex. 10 at 38. Petitioner underwent an x-ray of her hands on the same day, which revealed "[o]steoporosis[16] and osteoarthropathy."[17] *Id.* at 231. She returned to her PCP on November 16, 2015, with complaints of "pain in both hands[, e]specially at the base of the thumbs[, and s]ome numbness and tingling in the tips of the fingers." *Id.* at 36. Petitioner also complained of "localized pain [in her] left lateral ribs" beginning two or three days prior. *Id.* Petitioner's PCP diagnosed her with carpal tunnel syndrome ("CTS")[18] and prescribed "symptomatic treatment for

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[11] Degenerative joint disease is also known as osteoarthritis. *Dorland's* at 1326. For a definition of osteoarthritis, *see supra* note 9.

[12] Syncope is defined as "a temporary suspension of consciousness due to generalized cerebral ischemia . . . ." *Dorland's* at 1788. Ischemia is defined as a "deficiency of blood in a part, usually due to functional constriction or actual obstruction of a blood vessel." *Id.* at 949.

[13] A computed tomography scan is a "tomography in which the emergent x-ray beam is measured by a scintillation counter; the electronic impulses are recorded digitally and then are processed by a computer for reconstruction display." *Dorland's* at 1905. It is also known as a CT scan. *Id.* Tomography is "the recording of internal body images at a predetermined plane by means of the tomograph . . . ." *Id.* A tomograph is "an apparatus for moving an x-ray source in one direction as the film is moved in the opposite direction, thus showing in detail a predetermined plane of tissue while blurring or eliminating detail in other planes." *Id.*

[14] Lisinopril is "the lysine derivative of the active form of enalapril; an angiotensin-converting enzyme inhibitor used in the treatment of hypertension (alone or in combination with a thiazide diuretic), congestive heart failure, and acute myocardial infarction; administered orally." *Dorland's* at 1052.

[15] Hydralazine is "a peripheral vasodilator used as an antihypertensive." *Dorland's* at 865. Vasodilator is defined as "1. causing dilation of the blood vessels[; or] 2. a motor nerve or chemical compound that causes dilation of the blood vessels." *Id.* at 1996.

[16] Osteoporosis is defined as "reduction in bone mineral density, leading to fractures after minimal trauma." *Dorland's* at 1329.

[17] Osteoarthropathy is defined as "any disease of the joints or bones." *Dorland's* at 1326.

[18] Carpal tunnel syndrome is defined as "an entrapment neuropathy characterized by pain and burning or tingling paesthesias in the fingers and hand, sometimes extending to the elbow. Symptoms result from

[her] left rib pain." *Id.* at 37.

The next day, Petitioner returned to BARH hospital with complaints of "upper back pain[that] . . . prevents her from getting sleep . . . ." Pet'r's Ex. 4 at 74–75. She described the pain as "sharp in nature" and "rated [it eight-to-nine out of ten]" on the pain scale. *Id.* at 75. Her physical exam was normal, and Petitioner did not have any motor or sensory deficits. *Id.* at 76. Doctors diagnosed Petitioner with postural lower back pain and discharged her home. *Id.*

On November 23, 2015, Petitioner returned to her PCP with complaints of "[b]ack pain, left rib pain, [and] numbness [in] both [of her] hands." Pet'r's Ex. 10 at 34. Petitioner described "pain in [the] mid[-]thoracic spine area and to the left[,]" as well as "pain in [her] left ribs[, e]specially" in the "T7-T8 range." *Id.* Petitioner's PCP reviewed Petitioner's prior x-rays and noted "[s]ome osteoporosis and osteoarthritis[,]" and referred her for another NCS, an MRI of her thoracic spine, and an x-ray of her left ribs. *Id.* at 34–35. Petitioner also received a Lortab[19] prescription to manage her pain. *Id.*

Petitioner again presented to the emergency department at BARH hospital on November 24, 2015, for an "[i]njury to [her] upper back and thoracic spine." Pet'r's Ex. 4 at 55. She complained of "upper back pain[that was] sharp[and] radiate[d] to [the] anterior chest wall . . . ." *Id.* at 56. She stated that the pain was "worse with sitting up or laying down." *Id.* Repeat chest x-rays showed "no evidence [of an] acute cardiopulmonary process" or a "displaced left rib fracture." Pet'r's Ex. 10 at 335, 337. Petitioner had a normal physical examination, including a normal neurological examination with "no motor . . . [or] sensory deficit[s]." Pet'r's Ex. 4 at 56–57. Doctors again discharged Petitioner home with diagnoses of low back pain and chronic back pain. *Id.* at 58.

Petitioner had an appointment with Thomas Kowalkowski, M.D., a urologist, on November 30, 2015, for a chief complaint of blood in her urine. Pet'r's Ex. 5 at 39. Her active problems list included "flaccid neuropathic bladder"[20] and "urinary retention." *Id.* The HPI section of the note states that Petitioner presented for a "consultation regarding hematuria."[21] *Id.* Dr. Kowalkowski noted that Petitioner had "[n]o change in urinary frequency and no incomplete emptying of [her] bladder." *Id.* at 40. He also noted that Petitioner did not have any "dysuria."[22] *Id.* A urinalysis revealed +4 hemoglobulin in Petitioner's urine. *Id.* Dr. Kowalkowski assessed Petitioner with a "microscopic hematuria" and ordered renal and pelvic ultrasounds, which were unremarkable. *Id.*; *see also id.* at 96–97.

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compression of the median nerve in the carpal tunnel." *Dorland's* at 1794.

[19] Lortab is the "trademark for combination preparations of hydrocodone bitartrate and acetaminophen." *Dorland's* at 1061. Hydrocodone bitartrate is "the bitartrate salt of hydrocodone, used as an analgesic and antitussive; administered orally." *Id.* at 867.

[20] Neuropathic bladder refers to "any condition of dysfunction of the urinary bladder caused by a lesion of the central or peripheral nervous system." *Dorland's* at 219. The central nervous system is "the part of the nervous system consisting of the brain and spinal cord." *Id.* at 1829. The peripheral nervous system is "the part of the nervous system consisting of nerves and ganglia outside the brain and spinal cord." *Id.* at 1832.

[21] Hematuria is defined as "blood . . . in the urine . . . ." *Dorland's* at 824.

[22] Dysuria is defined as "1. painful urination[; or] 2. any difficulty of urination." *Dorland's* at 579.

On December 1, 2015, Petitioner presented to Dr. Whitfield for a consultation. Pet'r's Ex. 9 at 5. Petitioner complained of "increased numbness and tingling in both hands . . . [that] began about three weeks ago[] . . . in the fingertips and . . . has progressed toward the palm." *Id.* She noted that the "[p]aresthesias affect[ed] her thumb, index finger[,] and middle finger more so than the ulnar two digits." *Id.* She explained that "[b]oth hands are equal, though the right hand began before the left." *Id.* Petitioner also stated that "she developed some paresthesias about the toes on both of her feet[,]" but did not state when this began. *Id.* She denied "any weakness in her upper or lower extremities." *Id.* Dr. Whitfield recounted Petitioner's recent back pain and hospitalizations, which resulted in a diagnosis of a "muscle strain." *Id.* His examination revealed "[d]ecreased sensation about the palmar aspect of the thumb, index finger[,] and middle finger bilaterally." *Id.* at 6. He also noted that Petitioner had normal range of motion ("ROM"), reflexes, and no motor deficits. *Id.* at 7. His assessment included "[b]ilateral [CTS] versus peripheral neuropathy[]" and "[l]ower extremity neuropathy of unclear etiology." *Id.* He also assessed "severe osteoporosis" and "upper back pain [that he] suspect[ed resulted from a] compression fracture given [Petitioner's] osteoporosis." *Id.* Dr. Whitfield ordered an MRI of Petitioner's lumbar spine, an EMG, and fit Petitioner with hand braces to attempt to control her CTS. *Id.* at 8.

Petitioner underwent an EMG study with Barry Vaught, M.D., on December 3, 2015. *Id.* at 37. Dr. Vaught described the results as "abnormal" and concluded that there was "electrophysiologic evidence for severe bilateral median mononeuropathy at the wrists, consistent with [CTS]." *Id.* at 38. He also wrote that there was "no evidence [of] ulnar neuropathy on either side or cervical radiculopathy on the right." *Id.* Petitioner's PCP concurred with this diagnosis after reviewing these results with Petitioner at an appointment on December 9, 2015, and directed Petitioner to follow-up with Dr. Whitfield. Pet'r's Ex. 10 at 31–32.

On December 22, 2015, Petitioner returned to Dr. Whitfield for a follow-up. Pet'r's Ex. 9 at 2. Petitioner "complain[ed] of pain about the bilateral hands, . . . paresthesias about both of her hands[,] and state[d] that she has decreased sensation and . . . feels like she is picking things up, but she is not." *Id.* Petitioner also "complain[ed] of some pain about the thoracic spine that radiates across the axilla region to her breast on the right side." *Id.* Dr. Whitfield's examination revealed "[d]ecreased sensation about the palmar aspect of the thumb, index finger[,] and middle finger bilaterally." *Id.* Dr. Whitfield's assessed Petitioner with severe bilateral CTS, lower extremity neuropathy of unclear etiology, and severe osteoporosis that he "suspect[ed caused a] compression fracture . . . ." *Id.* at 3. He planned on performing a "carpal tunnel release"[23] because this procedure would "give the nerve the best chance of survival[, but noted that] it is possible that permanent nerve damage has been done." *Id.* He also referred Petitioner to physical therapy ("PT") for her back pain. *Id.*

Petitioner again returned to the emergency department at BARH hospital on December 29, 2015, "with a chief complaint of [right] foot and shin pain[]" caused by a fall at church the previous

[23] Carpal tunnel, or *canalis car´pi*, is defined as "an osseofibrous tunnel for passage of the tendons of the flexor muscles of the hand and digits, formed by the flexor retinaculum as it roofs over the concavity of the carpus on the palmer surface . . . ." *Dorland's* at 274. The carpus is "the joint between the forearm and hand, made up of eight bones . . . ." *Id.* at 293. A release is defined as a "surgical incision or cutting of soft tissue to bring about relaxation." *Id.* at 1597. It is not clear whether or when Petitioner underwent this procedure.

day. Pet'r's Ex. 4 at 43. Petitioner stated that she "bent over to pick up some plant leaves and when [she] raised up[,] [she] lost [her] balance[]" and fell. *Id.* An examination did not reveal any "obvious swelling, reddening, contusion, [or] deformity," and a neurological exam was normal. *Id.* Petitioner rated that pain "an [eight] on a [one-to-ten] pain scale." *Id.* Petitioner was diagnosed with an ankle injury and was noted to be "unsteady and at risk for multiple falls." *Id.* at 43–44.

On December 31, 2019, Petitioner reported to University of Virginia Hospital East.[24] Pet'r's Ex. 11 at 2; Pet'r's Ex. 6 at 463. Petitioner had an abnormal urinalysis, Pet'r's Ex. 11 at 2–3, as well as an abnormal complete blood count panel, Pet'r's Ex. 6 at 463. A lumbar spine MRI revealed "[n]o evidence of [an] acute fracture or other acute abnormality." Pet'r's Ex. 11 at 20–21. Petitioner was diagnosed with bilateral lower extremity pain and weakness and was discharged home with a prescription for oxycodone[25] and directions to follow-up with orthopedics. Pet'r's Ex. 6 at 463–64.

Petitioner presented to the emergency department at BARH hospital in a wheelchair on January 2, 2016, with complaints of "left side facial drooping and left extremity weakness" beginning one day prior. Pet'r's Ex. 4 at 33. Although Petitioner was "able to speak and answer questions[,]" doctors noted that Petitioner had a "left-sided facial droop[,]" left lower extremity weakness such that she was unable to "raise [her] left leg off [of the] bed[,]" and "[u]nequal hand grips with [the] left hand weaker[]" than the right. *Id.* Petitioner underwent a head CT, which was negative for stroke, *see* Pet'r's Ex. 10 at 483, and doctors diagnosed her with hemiplegia and slurred speech and transferred her to St. Mary's Hospital, Pet'r's Ex. 4 at 35.

Upon arrival at St. Mary's hospital, Petitioner continued to have "[l]eft-sided facial weakness[]" and "[d]ifficulty closing [her] right eye[,]" as well as moderate weakness in both legs and feet. Pet'r's Ex. 10 at 403. She related to her treating doctors "that she has had bilateral lower extremity [sic.] and difficulty walking since [December 29, 2015]." *Id.* at 438. Doctors wrote that Petitioner "has never had a finding of neurologic deficiency in either leg." *Id.* Petitioner underwent a "CTA of [her] head and neck[, which] showed no acute infarct[ion]."[26] *Id.* at 440. Doctors also reviewed Petitioner's previous lumbar spine MRI, which "did not show any significant disk abnormalities causing central canal stenosis." *Id.* Doctors also wrote that Petitioner's previous head CT did not reveal any "acute bleed." *Id.* at 441. The assessment included "[q]uestionable [transient ischemic attack ("TIA")][27] versus Bell's palsy" and "[d]ifficulty with ambulation secondary to pain." *Id.* Petitioner was admitted for a neurology consultation. *Id.*

Petitioner had an initial consultation with Ijaz Ahmad, M.D., a neurologist, on January 3,

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[24] It is unclear what specific complaints compelled Petitioner to present to the emergency department on this date. *See* Pet'r's Ex. 11 at 2–22; *see also* Pet'r's Ex. 6 at 463–70.

[25] Oxycodone is "an opioid agonist analgesic derived from morphine." *Dorland's* at 1337.

[26] Infarction is defined as "1. infarct[; or] 2. the formation of an infarct." *Dorland's* at 923. An infarct is "an area of coagulation necrosis in a tissue due to local ischemia resulting from obstruction of circulation to the area, most commonly by a thrombus or embolus." *Id.* at 922. For a definition of ischemia, *see supra* note 11.

[27] A transient ischemic attack is defined as "a brief attack (from a few minutes to an hour) of cerebral dysfunction of vascular origin, with no persistent neurologic deficit . . . ." *Dorland's* at 175.

2016. Pet'r's Ex. 6 at 328. He wrote that Petitioner's left-sided facial droop had "resolved." *Id.* Dr. Ahmad's "motor examination reveal[ed] normal strength, [but Petitioner] had proximal weakness in the lower extremities at plus [four]" and was "areflexic in the lower extremities." *Id.* at 329. He also wrote that Petitioner's "[v]ibration [sensation was] moderate[ly] decreased[,]" and that she "was unable to stand by herself." *Id.* Dr. Ahmad's impression was [w]eakness in the lower extremities with paresthesia[,]" although he also noted that "[o]ne has to consider acute inflammatory polyradiculopathy." *Id.* He explained that "[a]t this point[, he was] uncertain how [acute inflammatory polyradiculopathy] is associated with weakness of the lower extremities . . . ." *Id.* He continued, "however, if indeed this is an acute inflammatory adenopathy[, then] facial weakness is not unusual accompanying." *Id.* He ordered blood tests, a NCS, and an EMG study. *Id.*

On the same day, Petitioner underwent an EMG study, which revealed "marked asymmetrical prolongation of the distal latencies, particularly of the left median[and] right ulnar nerve." *Id.* at 331. It also showed "reduced recruitment pattern in all of the muscles tested[,] which was more pronounced in the proximal muscles of the lower extremities[,]" as well as "evidence of loss of motor units and evidence of denervation pattern in the lumbar paraspinal muscles." *Id.* Dr. Ahmad wrote that "[t]hese findings are indicative of asymmetrical predominantly demyelinating neuropathy[,] which seems to be acute." *Id.* He ordered a five-day IVIG treatment, which Petitioner completed on January 9, 2016. *See* Pet'r's Ex. 6 at 27–28.

Petitioner "improved slightly" after the IVIG treatments, although "[s]he initially seemed to get worse and was a max assist." Pet'r's Ex. 10 at 318. However, by January 13, 2016, Petitioner was a "moderate assist and believe[d] that her legs [were] moving better." *Id.* During her hospitalization, Petitioner received both PT and occupational therapy ("OT") daily, *see* Pet'r's Ex. 6 at 408–10, but "continued to have a lot of pain difficulties[,]" Pet'r's Ex. 10 at 318. She also experienced "significant urinary retention" issues, which doctors treated with a Foley catheter, and autonomic dysfunction, which doctors believed was "probably related to her neurologic condition." *Id.* at 317. Petitioner was discharged on January 13, 2016, with diagnoses including "[a]cute [GBS] per Dr. Ahmad[,]" "ambulatory dysfunction[,]" and "chronic back pain – disk disease on MRI." *Id.* at 317. She was transferred to HealthSouth for inpatient rehabilitation. *Id.*

Petitioner received intensive inpatient PT until February 1, 2016. *See* Pet'r's Ex. 7 at 191. During her stay, Petitioner developed pneumonia, which resolved prior to her discharge. *See id.* at 390. Upon discharge, Petitioner's functional status required "[s]upervision [with] eating and grooming[, moderate] assistance with bathing[, t]otal assist[ance] with lower extremity dressing[, and moderate] assist[ance] with toilet transfers." *Id.* She also continued to experience urinary retention issues and remained on a Foley catheter. *Id.*

On February 3, 2016, Petitioner was readmitted to BARH hospital for a "catheter problem." Pet'r's Ex. 4 at 8. Emergency medical services reported that Petitioner was "having severe lower abdominal pain due to a possible blocked Foley catheter." *Id.* Petitioner's neurological examination was normal with no motor or sensory deficits. *Id.* at 10. Petitioner's primary diagnoses were "[u]rinary tract obstruction[,]" "[l]ower urinary tract symptoms[,]" and "[l]ower urinary tract infectious disease." *Id.* at 11. She was prescribed antibiotics and discharged home. *Id.* at 12.

Petitioner presented to Dr. Kowalkowski for a consultation on February 5, 2016. Pet'r's Ex. 5 at 31. Dr. Kowalkowski wrote that Petitioner "was gradually getting strength back in her lower extremities but is unable to walk on her own yet." *Id.* at 33. He also advised Petitioner to "start [PT] for lower extremity strengthening[,]" as "[t]he quicker she is able to walk the better her bladder function is going to be." *Id.* Dr. Kowalkowski kept Petitioner's Foley catheter in place and wrote an order for home health care to change it once a month. *Id.*

Petitioner attended her first outpatient PT session on February 16, 2016, at Athletic & Physical Therapy Services. Pet'r's Ex. 3 at 1. The physical therapist wrote that there was "difficulty [with diagnosing GBS] and [Petitioner] was seen by several doctors before finally having the [diagnosis]." *Id.* Petitioner had "good sitting balance but very poor standing balance and [was a] max assist [on] transfers." *Id.* She "also need[ed] assist[ance] with rolling on [a] table and [moving from] supine to sitting." *Id.* Petitioner had full ROM in her hips, knees, and ankles, but had decreased strength in each. *Id.* She was assessed as having "[very] weak mid[-]trunk and hips [but] better with quads and ankles[,]" as well as "severe atrophy of hips and quads." *Id.* at 2. The plan was for Petitioner to "be seen in [the] clinic [two-to-three] days a week for [four-to-six] weeks . . . ." *Id.* Petitioner attended approximately eighteen PT sessions through April 6, 2016. *See id.* at 3–20. At her last session, Petitioner showed improved strength and was "able to lower [herself] from [an] elevated position but [was] not [able] to raise for [sic.] flexed position . . . ." *See id.* at 20.

On April 13, 2016, Petitioner underwent a repeat EMG study. Pet'r's Ex. 10 at 347. The results revealed "moderate prolongation of distal motor latencies in the upper extremities[and] . . . mild-to-moderate prolongation of distal motor latencies on the peroneal nerve." *Id.* The test also showed "decreased recruitment pattern, particularly in the lower extremities[,]" and "some denervation potential in the right vastus medialis and lumbar paraspinal muscles." *Id.* The interpretation was "further slowing of the conduction velocities as compared to the previous exam on January 2, 2016. Minimal denervation potentials were seen in the right quadriceps and lumbar paraspinal muscles." *Id.*

Petitioner had a follow-up visit with Dr. Ahmad on May 17, 2016. Pet'r's Ex. 12 at 2. Petitioner complained of dizziness occurring "since [she was] discharged from [the] hospital." *Id.* She described it as "intermittent" and occurring "often." *Id.* Petitioner also noted general weakness that had improved with PT. *Id.* Petitioner complained of "episodes when [her] vision [became] 'weak' and she [had] to blink her eyes to focus." *Id.* On examination, Dr. Ahmad noted "only minimal weakness along the proximal muscles in the lower extremities. Deep tendon reflexes [("DTRs")] are 1+. Plantar responses are flexors. Sensory examination is unremarkable." *Id.* Dr. Ahmad also wrote that Petitioner's dizziness "seems to be [of] peripheral etiology." *Id.* at 3. He advised Petitioner to see an ear, nose, and throat doctor and directed her to follow-up with him in two-to-three months. *Id.* at 2–3.

On June 29, 2016, Petitioner was readmitted to BARH hospital for "dizziness" that began "approximately [three] weeks ago[,]" but had been "intensifying since . . .[six] days prior to presentation." Pet'r's Ex. 13 at 11. Petitioner reported that she had recently been diagnosed with

"benign paroxysmal positional vertigo."[28] *Id.* Petitioner also reported "intermittent chest pain episodes for the past week at rest[]" with "palpitations [that] can last for several minutes in duration." *Id.* She described "get[ting] dizzy whenever she ben[t] forward to brush her teeth, and [when she] sits back up and [into a] standing position[, she] becomes very lightheaded." *Id.* The doctor also wrote that Petitioner should "[a]void flu vaccination as flu vaccination exacerbated [GBS]." *Id.* at 14. Petitioner was admitted for further evaluation. *Id.* at 12. She was discharged on July 1, 2016, with diagnoses including "[d]izziness, vertigo, multifactorial related to allergic rhinitis[,]" and "[a]typical chest pain in the absence of myocardial infarction, most likely musculoskeletal in origin." *Id.* at 26.

B. Affidavits

1. Petitioner's Affidavit

Petitioner filed her affidavit on December 19, 2017. Pet'r's Ex. 16. Petitioner explained that approximately four weeks after receiving the flu vaccination at issue, she "noticed the emergence of adverse symptoms," including "malaise that left [her] run-down and tired[]" and "a tingling sensation in [her] fingers and toes[,]" which "transitioned into general muscle weakness." *Id.* at 1. She stated that the "weakness progressed, especially in [her] legs, and resulted in a fall while at church in late October . . . ." *Id.* After this fall, Petitioner wrote that she "fell again a few days later while at home." *Id.* She presented to her PCP "to figure out and address the[se] disturbing symptoms[,]" but was discharged without a diagnosis. *Id.*

Petitioner wrote that she presented to the emergency room in "the early days of November 2015[,]" because her "symptoms continued to worsen[] and the pain in [her] hands grew . . . intolerable . . . ." *Id.* at 2. At the emergency room, Petitioner stated that her "blood pressure was noted to be very high," a finding that she feels could have been because she "was in a great deal of pain and discomfort" at that time. *Id.* Petitioner stated that "[a]ll the testing they performed on [her] was unable to reveal a cause for [her] symptoms, so [she] was again discharged home." *Id.*

After another couple of weeks, Petitioner wrote that she "was back in the emergency department" because her "symptoms grew even worse." *Id.* She again noted that testing could not reveal a cause of her symptoms, and after a couple of days, she was discharged home. *Id.* Petitioner stated that "[t]his pattern continued a couple of more times and [she] sought help at the University of Virginia Hospital on [December 31, 2015] . . . ." *Id.* She explained that "[b]y the time [she] arrived there, [she] could no longer walk at all due to the pain and weakness in [her] legs." *Id.*

On January 2, 2016, Petitioner stated that she returned to BARH hospital because her "pain . . . had progressed to the point of being unbearable," and she was suffering from "weakness in [her] arms and legs, and a drooping mouth." *Id.* Petitioner explained that she was transferred to St. Mary's Hospital "once the emergency doctors agreed that [her] symptoms seemed to have a neuropathic aspect." *Id.* Petitioner noted that "a lumbar puncture . . . led Dr. Ahmed to determine

[28] Petitioner reported that she had been diagnosed with this condition by a "Dr. Payne." Pet'r's Ex. 13 at 11. However, it is unclear when this diagnosis was made or where this diagnosis is located in Petitioner's filed medical records.

that [she] had been suffering from [GBS]," and she was "started on treatments and . . . stayed at St. Mary's for two weeks." *Id.*

After her discharge from St. Mary's Hospital, Petitioner wrote that she was transferred to inpatient rehabilitation at HealthSouth, which "last[ed fifteen] hours a day and extend[ed] through the end of February 2016." *Id.* at 3. Despite noting that she "made very little progress" during her inpatient stay, Petitioner stated that she "had to leave the hospital . . . to return home." *Id.* Upon discharge, Petitioner wrote that she "was an emotional wreck[]" because she left rehab "with deep fear and depression with the realization that [she] might never walk again, that [she] would need [twenty-four]-hour care, and that [she] would remain unable to hear the world around [her]."[29] *Id.*

Petitioner explained that, "[t]hroughout the period from early December [2015] through early March [2016], the pain was so unbearable that Oxycontin was insufficient to give [her] comfort." *Id.* In addition, she noted that "[t]he urinary catheter brought on an infection for which [she] needed antibiotics, but which became recurrent and even more painful over time." *Id.* During this period, Petitioner wrote that she "was crying daily and really struggling with depression." *Id.* Also, during this time period, Petitioner stated that she "was thoroughly disabled and living at home, [so her] daughter Maria and [her] oldest son Frank were [her] primary caregivers and . . . did everything for [her] . . . ." *Id.*

In late March of 2016, Petitioner stated that her "condition finally started to improve somewhat . . ., but certainly did not resolve." *Id.* She wrote that the "[urinary tract infections ("UTIs")] would not go away, and [she] developed an[] allergic response to the antibiotics [she] was prescribed to treat it." *Id.* In addition, she stated that the "urologist conditioned [the] removal of the extremely uncomfortable catheter upon [her] being able to walk . . . ." *Id.* Petitioner wrote that she only "had to return to the hospital . . . to receive intravenous antibiotics." *Id.*

Petitioner stated that she "regained the ability to stand in late April 2016." *Id.* at 4. She also noted that her "pain was becoming manageable if not tolerable" at this time as well. *Id.* Petitioner stated that she "began to relearn how to walk" and had the catheter removed in early May 2016. *Id.* However, she wrote that "[i]t was late August [2016] before [she] was able to walk unassisted, albeit quite slowly." *Id.* She concluded her affidavit by noting that "[t]he entire affair has been a constant traumatic experience[]." *Id.*

2. Affidavit of Maria Fly

Petitioner also submitted an affidavit from her daughter, Maria Fly, on December 19, 2017. Pet'r's Ex. 17. Ms. Fly stated that "[a]bout one week after [Petitioner was] administered a flu vaccination . . ., [Petitioner] began to complain that she felt funny[and that] she did not feel right." *Id.* at 1. Ms. Fly noted that she and her siblings "at first dismissed [Petitioner's] complaints[]" because Petitioner "could not pinpoint the exact nature of what she was feeling . . . ." *Id.* Ms. Fly further noted that she and her siblings "had always understood that the flu shot can cause flu-like symptoms . . . ." *Id.* However, Ms. Fly explained that "[o]ver the weeks that followed, . . . [Petitioner] began expressing more specific complaints of pain in her back, her hands, and her feet,

[29] Petitioner also stated that "the staff at the rehab hospital lost [her] hearing aids and they were never found." Pet'r's Ex. 16 at 3.

which became progressively worse." *Id.* Ms. Fly recounted the "prolonged process of bringing [Petitioner] to the doctors for diagnosis and treatment, only to be sent away without answers." *Id.* She noted that "[s]oon [Petitioner's] pain was joined by numbness and tingling in her hands and feet that was so uncomfortable that she could not rest." *Id.* Ms. Fly wrote that this pain "was also putting stress on [Petitioner's] body, elevating her blood pressure to dangerous levels . . . ." *Id.* Ms. Fly also noted that Petitioner's "pain at night was horrific[] and stole her sleep from her." *Id.* at 2. Ms. Fly wrote that she has "videos of [Petitioner] rolling and thrashing in an attempt to relieve her pain and try to sleep." *Id.* Ms. Fly also explained that "[t]here were many instances where [Petitioner] would not sleep for more than [thirty] minutes all night, and only catch a [fifteen] minute nap during the day . . . ." *Id.*

On December 27, 2015, Ms. Fly stated that Petitioner "fell while attending church[,]" and in light of her previous symptoms, Ms. Fly and her brother "decided to take [Petitioner] to the University of Virginia hospital a few days later." *Id.* However, Ms. Fly stated that the doctors "had the same ineffectual response to [Petitioner's] extreme condition, except to guess about arthritis in [Petitioner's] back." *Id.* Ms. Fly recounted that Petitioner was discharged home "with highly elevated blood pressure and no relief from her pain and sensory discomfort." *Id.*

Ms. Fly continued that on January 2, 2016, she and her brother "took breakfast to [Petitioner's] house" where Ms. Fly "first noticed a severe droop affecting the entire right side of [Petitioner's] face." *Id.* Ms. Fly also noted that Petitioner "had very little control over the opening and closing of her eyelid, and her mouth drooped so much[that] she was drooling noticeably." *Id.* In addition, Ms. Fly recounted that Petitioner "could not walk at all." *Id.* Because of Petitioner's condition, Ms. Fly and her brother "rushed [Petitioner] to [the] local emergency room . . . ." *Id.* After doctors transferred Petitioner to St. Mary's Hospital, Ms. Fly noted that Petitioner was diagnosed with GBS and "began an eleven-day stay at the hospital." *Id.* During this time, Ms. Fly stated that Petitioner "could not walk and . . . was heavily medicated in an effort to ease her pain and lower her dangerously elevated blood pressure." *Id.* Ms. Fly stated that she remained in the hospital with Petitioner "around the clock" for a majority of Petitioner's time at St. Mary's Hospital. *Id.* at 3.

After Petitioner's inpatient rehabilitation, Ms. Fly stated that Petitioner's "body had become so weak that she could not walk or stand, and her bladder was not working properly, requiring that she wear a continuous catheter." *Id.* Ms. Fly recounted that Petitioner's weakness was so profound that Ms. Fly or her brother "had to pick [Petitioner] up to move her from one place to another." *Id.* Ms. Fly noted that Petitioner's "sensory problems in her hands[]" made it so that she "could barely grasp anything and could not hold onto anything . . . ." *Id.* Therefore, Ms. Fly "got [Petitioner] a children's 'fat-handle' spoon and fork set so she could try and feed herself." *Id.* Ms. Fly explained that, "from February into March of 2016," she "was the one primarily responsible for [Petitioner's] personal care and for cooking [Petitioner's] meals" because Petitioner "was reduced to an invalid." *Id.*

Ms. Fly also stated that, once Petitioner was home from inpatient rehabilitation, "a home-health therapist came to her house at first, but soon determined that [Petitioner] needed to attend outpatient [PT] so she could use the exercise machines to strengthen her atrophied muscles, which she began to do about three weeks after returning home." *Id.* These PT sessions "were an hour or

more long, and typically convened three times a week." *Id.* at 3.

Ms. Fly also discussed Petitioner's catheter and UTIs, which Ms. Fly described as "a source of pain for [Petitioner] and stress for all of [Petitioner's family]." *Id.* at 4. Ms. Fly explained that "[w]henever [she] took [Petitioner] outside, [Ms. Fly] had to change [Petitioner's] catheter bag to a travel-sized bag." *Id.* Ms. Fly also noted that Petitioner eventually required intravenous antibiotics to treat her recurrent UTIs, which required hospitalization. *Id.* Even after the catheter was removed, Ms. Fly stated that Petitioner "had to retrain her bladder[]" and "began wearing adult diapers and still does today." *Id.*

3. Affidavit of Frank Conte

Petitioner submitted a third affidavit, from her son, Frank Conte, on December 19, 2017. Pet'r's Ex. 18. Mr. Conte stated that he "noticed several days following [Petitioner's flu] vaccination . . . that she was just not herself." *Id.* at 1. Specifically, he wrote that Petitioner "began to complain of pain in her hands, back, and feet[,]" which "was soon disturbing her sleep." *Id.* Mr. Conte recounted that Petitioner "would pace through the house looking for a place to lie down and get comfortable despite her discomfort and pain." *Id.*

Mr. Conte noted that he and his sister "brought [Petitioner] to doctors and the hospital on several occasions, but they could not discover the cause of her problems," all "while her pain became unbearable for her . . . ." *Id.* In addition to her pain, Mr. Conte stated that Petitioner experienced "weakness and instability, and . . . she fell a couple of times . . . ." *Id.* Mr. Conte noted that, once Petitioner "developed the facial palsy[,] . . . she was seen by a neurologist" and diagnosed with GBS. *Id.* at 2. However, Mr. Conte explained that by the time of diagnosis, Petitioner "could not walk, and she could hardly use her hands at all. She was in constant, extreme pain." *Id.* Mr. Conte further noted that, "[a]fter eleven days at St. Mary's Hospital and seventeen days at HealthSouth [inpatient rehabilitation, Petitioner] was discharged home, but . . . [still] could not walk or stand, and could barely hold utensils to eat." *Id.*

While Petitioner was recovering at her home, Mr. Conte stated that he "had to do everything she needed done, whether to lift her onto the toilet, help her with other hygiene, to administer her medications, and to help her dress." *Id.* In addition, Mr. Conte explained that he "carried [Petitioner] into and out of the car for all of her many medical appointments in the months after hospital discharge." *Id.* Mr. Conte also stated that he "became responsible to do all grocery shopping, cleaning, laundry and related chores[]" for Petitioner during this time period. *Id.* He concluded by explaining that, while Petitioner "did slowly improve and regain some strength[,]" he nonetheless "still bear[s] most of the responsibilities around the house." *Id.* at 3.

III. Experts

A. Expert Qualifications

1. Petitioner's Expert, Zurab Nadareishvili, M.D., Ph.D.

Dr. Nadareishvili received his medical and doctoral degrees from the Tbilisi State Medical

Institute in Tbilisi, Republic of Georgia in 1984 and 1991, respectively. Pet'r's Ex. 20 at 1. He is board certified in neurology and vascular neurology and is licensed to practice medicine in Maryland and the District of Columbia. *Id.* at 2. He currently serves as the Medical Director of the National Institute of Health ("NIH") Stroke Program and the Chair of the Subsection of Neurology, Department of Medicine at Suburban Hospital, which is part of Johns Hopkins Medicine in Bethesda, Maryland. *Id.* at 1. He also serves as a faculty member in the Vascular Neurology Clinical Fellowship program at the National Institute of Neurological Disorders and Strokes within the NIH in Bethesda, Maryland, and as an Associate Clinical Professor of Neurology at the George Washington University School of Medicine and Health in Washington, D.C. *Id.* His curriculum vitae lists twenty-eight peer-reviewed articles of which he is a listed author, as well as one book chapter and forty-one abstracts. *See id.* at 10–17. Dr. Nadareishvili submitted three expert reports in this case. *See* Pet'r's Exs. 19, 38, 44.

2. Respondent's Expert, Vinay Chaudry, M.D.

Dr. Chaudry received his Bachelor of Medicine and Bachelor of Surgery from the All India Institute of Medical Sciences in New Delhi, India, in 1980. Resp't's Ex. B at 2. He currently serves as a Professor of Neurology at Johns Hopkins University School of Medicine and as the Director of the EMG Laboratory at Johns Hopkins Hospital. Resp't's Ex. A at 1. He holds board certifications in neurology, neuromuscular diseases, electrodiagnostic medicine—specifically in NCSs and EMGs—and clinical neurophysiology. *Id.*; *see also* Resp't's Ex. B at 36. He wrote in his first expert report that he has "an active clinical practice" where he "evaluate[s] 2,000 patients a year[,] mostly related to peripheral nerve disease." Resp't's Ex. A at 1. His curriculum vitae lists over one hundred and fifty peer-reviewed articles, book chapters, and other publications of which he is a listed author. *See* Resp't's Ex. B at 3–14. Dr. Chaudry submitted three expert reports in this case. *See* Resp't's Exs. A, D, L.

B. Expert Reports

1. Dr. Nadareishvili's Expert Reports

In his first expert report, Dr. Nadareishvili described GBS as "a life-threatening, acute, monophasic, autoimmune, peripheral neuropathy." Pet'r's Ex. 19 at 8. He noted that, "in its classic form[, GBS] presents as a clinical syndrome of progressive, ascending numbness, weakness, and (potentially) paralysis, affecting the lower extremity primarily, but often affecting the upper extremity and, in severe cases, affecting the sensory and motor nerves of the torso." *Id.* Dr. Nadareishvili wrote that, "[w]ithin North America, [GBS] is typically understood to result from [a]cute [i]nflammatory [d]emyelinating [p]olyneuropathy . . . ("AIDP"), in which an immune-mediated process causes damage to the myelin sheathing of the peripheral nerves at or near their root junctions . . ., and can result in damage to the nerve axons themselves." *Id.* He also noted that, "[w]hile the hallmark of the syndrome is that it is monophasic, the recovery curve . . . is variable, leaving some with more complete recoveries and others with very significant residual deficits." *Id.*

Along with his first expert report, Dr. Nadareishvili submitted a paper by Willison et al., in which the authors provide a more detailed discussion of GBS. S*ee* Pet'r's Ex. 33.[30,31] They state that "[GBS] is usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots." *Id.* at 1. In the AIDP form of GBS, the "immune injury specifically takes place at the myelin sheath and related Schwann-cell components . . . ." *Id.* at 3. Clinically, the authors note that "rapidly progressive bilateral weakness is the key presenting symptom in most patients." *Id.* at 5. This weakness is "classically described as ascending, and usually starts in the distal lower extremities, but can start more proximally in the legs or arms." *Id.* They note that patients may also initially experience "sensory signs, ataxia, and features of autonomic dysfunction." *Id.* The authors also wrote that "[m]uscle pain or radicular pain, often but not always in the spinal region, is another frequent initial sign, which can complicate the diagnosis because pain can proceed weakness in about a third of patients." *Id.* They explain that "[m]ost patients . . . reach the nadir [of weakness] within [two] weeks[,]" although "[p]rogression can last up to [six] weeks after onset (subacute [GBS]) in some rare cases." *Id.* In terms of patient outcomes, the authors state that "[t]he severity and duration of the disease is highly diverse in patients and can range from mild weakness . . . to patients becoming quadriplegic and ventilator dependent . . . ." *Id.* They also note, however, that "all patients start improving [eventually], although recovery could follow a protracted course and result in severe, permanent disability." *Id.*

The authors of the Willison et al. paper also provide a panel in which they list the diagnostic criteria for GBS. *Id.* The authors list "[p]rogressive weakness in [the] legs and arms (sometimes initially only in the legs)" and "[a]reflexia (or decreased tendon reflexes in weak limbs)" as the two "[f]eatures needed for diagnosis of [GBS] in clinical practice[.]" *Id.* They also note that NCSs "[c]an be helpful in clinical practice, but are generally not required to diagnose [GBS]." *Id.*

Dr. Nadareishvili then wrote that the "[s]tructural similarities between microbial and self-peptides can result in the activation of autoreactive T cells[, which] has been referred to as 'molecular mimicry.'" Pet'r's Ex. 19 at 8–9. He explained that "[i]n a classical molecular mimicry scenario, antibodies cross-react directly with viral and cellular proteins . . . ." *Id.* at 9. He further explained that "[t]he causative process for this inflammatory destruction of the myelin on the peripheral nerves is most commonly described as being initiated by cross-reactivity of certain immune cells, from an initial immunologic response to an immune challenge . . ., and transferring to inadvertent immune reaction against self cells." *Id.* at 8. He listed "a virus, a bacterium, or a vaccine" as examples of an immune challenge that could trigger this inflammatory process. *Id.*

However, Dr. Nadareishvili also wrote that "[m]olecular mimicry is but one of several potential mechanisms by which an auto-immune [sic.] reaction to a foreign antigen can ultimately result in nerve demyelination." *Id.* at 9. He explained that "there is also a well-established model of T-cell activation to cross-reaction against myelin basic protein[,]" which could "damage nearby cells, leading to collaterally damaging effects to cell structures, both by release of cytokines and by macrophage activation." *Id.* He also discussed the "fertile field model," whereby "activation of an immune response by an immunogen with sequence homology to self proteins may prime autoreactive T-cells in the host, but might lack the momentum to initiate an adverse auto-immune

[30] Hugh J. Willison et al., *Guillain-Barré Syndrome*, 388 LANCET 717 (2016).
[31] Respondent also filed this article in support of Dr. Chaudry's second expert report. *See* Resp't's Ex. G.

[sic.] reaction independently." *Id.* He continued, "[h]owever, a later immune stimulation or coincidental immune stimulation event by a vaccine or other immunogen, even one without cross-reactive antigens or sequence similarity, could initiate an autoimmune reaction in a susceptible host that could lead to inflammatory demyelination." *Id.* He also briefly discussed bystander activation, noting that "[c]omponents of the vaccine could facilitate aberrant immune responses against self-antigens by binding to specific stimulating receptors to hyperactivate immune cells to overcome self-tolerance . . . ." *Id.* at 11–12.

In his first expert report, Dr. Nadareishvili opined that GBS can be caused by vaccination via molecular mimicry. *Id.* at 9. He argued that "[i]t is well established that vaccines interact with [the] innate and adaptive immune system[s] to boost reactivation of memory B cells generated during primary vaccination." *Id.* at 11. He explained that this interaction "is characterized by the rapid increase of higher titers of antibodies that will have a higher affinity for the target antigen(s) than those antibodies generated during primary vaccination to establish or boost vaccine specific immunity." *Id.* He then articulated how a vaccine could spur molecular mimicry: "the inactivated vaccines are taken up by antigen processing cells, which then breakdown the component proteins into small peptides prior to presentation to B and T lymphocytes." *Id.*

Dr. Nadareishvili argued that "[a]ll objective, diagnostic evidence in [Petitioner's] medical record supports . . . the diagnosis of AIDP-GBS over and above any other potential diagnosis." *Id.* at 13. He noted that any "competing diagnoses . . . were abandoned by most of [Petitioner's] treating physicians once objective evidence confirmed" this diagnosis in January 2016. *Id.* In addition, he wrote that Petitioner's course was "monophasic, without any fluctuating pattern that would suggest chronic inflammatory demyelinating polyneuropathy [("CIDP")]." *Id.* at 13–14.

Dr. Nadareishvili disagreed with Respondent's contention that Petitioner's first manifestation of symptoms occurred in late December 2016. *Id.* at 14; *see also* Resp't's Report at 11. He instead noted that "lower extremity neuropathy . . . was present at least as early as [Petitioner's] first visit to the orthopedist, Dr. Whitfield, on December 1, 2015." Pet'r's Ex. 19 at 14 (citing Pet'r's Ex. 9 at 5).

In addition, Dr. Nadareishvili disagreed with Respondent's argument that Petitioner's "symptoms and complaints within the three to forty-two[-]day period post-vaccination were clearly attributed by her doctors to CTS and her pre[-]existing hypertension and chronic back pain." *See id.* at 14–15 (quoting Resp't's Report at 12). Dr. Nadareishvili described CTS as "typically a chronic condition associated with repetitive use that progresses over time, and it is usually asymmetric in presentation, dependent on which hand is used more in the manner that aggravates the nerve." *Id.* at 15. He wrote that CTS does not "typically involve[] . . . the ulnar nerve." *Id.* He also wrote that, in severe cases of CTS, there is an "expect[ation] of a limitation in wrist mobility due to inflammation of the connective tissues . . . ." *Id.*

Dr. Nadareishvili argued that Petitioner did not fit the "clinical presentation" of CTS. *Id.* He began this discussion by noting that Petitioner "suffered a rather abrupt onset of sharp pain that was bilateral, and nearly symmetric in distribution between her hands." *Id.* Because Petitioner is retired, Dr. Nadareishvili found it "difficult to imagine that [Petitioner] would be performing some new movement that would cause such an acute flare up of nerve pain . . . ." *Id.* He also wrote that

"there was no evidence that [Petitioner's] wrist mobility was affected[,]" and the wrist splints prescribed by Dr. Whitfield, "a typical and effective treatment for [CTS,] had no effect in alleviating [Petitioner's] symptoms . . . ." *Id.* In addition, Dr. Nadareishvili noted that Petitioner's EMG results "showed involvement of both the median and ulnar nerves of the hand[,]" a finding which Dr. Nadareishvili believed was "glossed over in service to confirming the CTS diagnosis for which [Petitioner] was referred." *Id.* at 15–16. He continued, "[t]he testing is clear that [Petitioner's] neuropathy was severe, which would be quite unexpected with a chronic condition like [CTS]." *Id.* at 16. Dr. Nadareishvili noted that Dr. Ahmad, Petitioner's neurologist who made the GBS diagnosis, "did not support [a] diagnosis of [CTS,]" noting that "[e]ven in follow-up appointments . . ., [Dr. Ahmad] never mentioned" a CTS diagnosis. *Id.*

Rather, Dr. Nadareishvili believed that, "taken together, [Petitioner's] symptoms show a progressive course of immune-mediated neuropathy." *Id.* at 19. He discussed four symptoms that he described as diagnostic of GBS. First, he noted that the "first reference to . . . pain and paresthesias in [Petitioner's] lower extremities was in Dr. Whitfield's notes following [Petitioner's] December 1, 2015 initial visit . . . ." *Id.* at 16. He opined that "[t]his finding would be characteristic of [GBS] . . . ." *Id.* at 16–17. Second, Dr. Nadareishvili noted that Petitioner's "lower extremity weakness, . . . reduced grip strength, and . . . facial drooping are all clearly manifestations of [GBS] . . . ." *Id.* at 19.

Third, Dr. Nadareishvili discussed "the torsional pain, which [Petitioner] consistently reported, even if [doctors] inconsistently described and diagnosed." *Id.* at 17. He noted that Petitioner first complained of this pain on November 2, 2015, when she "reported . . . chest pain that radiated to [her] back[that] was severe enough to wake her from sleep." *Id.* Doctors "sent [Petitioner] home with recommendations for treatment of hypertension." *Id.* Dr. Nadareishvili then briefly reviewed the numerous visits Petitioner had for torsional pain over the course of the next month, including a visit with Dr. Whitfield on December 1, 2015, during which Dr. Whitfield "presume[d] a compression fracture must be the cause." *Id.* at 17–18. Dr. Nadareishvili argued that Petitioner's complaints "do not conform to an orthopedic, spinal, or cardiac explanation, and, indeed, no objective evidence exists in the record to support these as the source of [Petitioner's] pain." *Id.* at 18. Instead, Dr. Nadareishvili believed that they are "much more consistent with widespread, polyneuropathic pain, as the pain would be more locally limited if it arose from a spinal injury or cardiac condition." *Id.* He noted that "there are case reports and general acceptance of thoracic, abdominal, and back pain as acceptable (albeit atypical) presenting symptoms for [GBS]." *Id.* He continued, "[t]he pain in and around the back in particular represents the ongoing radiculopathic process of AIDP, which typically presents with distal symptomatology even when the neuropathy is more proximal to the spinal cord." *Id.* He also cited to medical articles accompanying his first report to support the proposition that there are "GBS cases which presented first with pain in the torso[,] more typically in the lower back, but also in the abdomen and upper back." *Id.*

Dr. Nadareishvili submitted a case report by Wong et al., where the authors "report a [GBS] case in which abdominal pain was the patient's initial symptom and her main complaint." Pet'r's Ex. 34 at 1.[32] The authors noted that the patient, a nineteen-year-old woman, presented with "a

[32] P.S. Wong et al., *Abdominal Pain as a Presenting Symptom of the Guillain-Barré Syndrome*, 5 ARCHIVES OF EMERGENCY MED. 242 (1988).

[three]-week history of stabbing pains in the right side of her abdomen." *Id.* The patient described the pain as "last[ing] for minutes or several hours, occur[ring] once every [two] days[,] and sometimes woke her up from sleep." *Id.* The patient also related that "[t]he pains were relieved by moving about." *Id.* Three days before the patient presented to the hospital, "she noticed similar shooting pains in both legs and some weakness when walking upstairs," and "[o]n the day of admission, she fell to the floor when she tried to get out of bed." *Id.* When she arrived at the hospital, she stated that "her legs felt weak and painful but the abdominal pain was the symptom which she emphasized (in spite of her weakness)." *Id.* at 2. On exam, the patient had "absent [tendon reflexes] in the legs but with reinforcement there was a patchy preservation in the arms[,]" and "the patient was not able to walk unaided." *Id.* The patient was ultimately diagnosed with GBS after an EMG study. *Id.* The only conclusion the authors make in the case report is that "[i]t is . . . important to diagnose [GBS] early[,]" and they expressed a desire to "draw attention to abdominal pain being a presenting symptom[]" of GBS. *Id.* at 3.

Fourth, Dr. Nadareishvili "believe[s] that [Petitioner] was experiencing autonomic dysfunction as a part of her onset of [GBS]." *Id.* at 18. He noted that Petitioner "did not have a history of autonomic dysfunction prior to her vaccination . . . ." *Id.* However, Petitioner developed "uncontrolled hypertension that resulted in syncopal episodes" as well as "urinary retention" post vaccination. *Id.* at 18–19. Dr. Nadareishvili argued that "[a]utonomic dysregulation and dysfunction can certainly signal the onset of [GBS]." *Id.* at 19. Along with his first expert report, Dr. Nadareishvili submitted a case report by Ferraro-herrera et al., which documents a sixty-one-year-old male GBS patient whose presenting symptom was hypertension. Pet'r's Ex. 36 at 1. The patient had "a history of mild hypertension" and "was admitted to the coronary care unit because of three episodes of chest pain while at rest and bifrontal headache." *Id.* He "also reported poorly controlled blood pressure . . . for a few days prior to admission." *Id.* The patient had a normal initial evaluation with a normal neurological examination. *Id.* However, on "the second day after admission, the patient began to develop progressive gait ataxia followed by the onset of bilateral proximal lower extremity weakness, areflexia, and numbness on the palms and soles of his feet." *Id.* He was diagnosed with GBS based on a cerebral spinal fluid ("CSF") study. *Id.* The authors state that "[h]ypertension occurs in [sixty percent] to [seventy percent] of patients with GBS." *Id.* at 2. They also note that "[c]ardiovascular dysautonomy is often present in patients with extensive motor involvement, and may be present both during the late progressive phase of the disease and during the plateau phase." *Id.* The authors do not draw any conclusions, however, regarding hypertension as a presenting symptom of GBS, noting only that "[p]atients with new cardiovascular symptomology should be followed for any subsequent changes in their neurological examination that are suggestive of GBS." *Id.*

Dr. Nadareishvili conceded that Petitioner's "progression of symptoms falls outside the normative standards accepted by the [Vaccine Injury] Table criteria" for GBS. Pet'r's Ex. 19 at 19. However, he nonetheless had "no trouble describing [Petitioner's] syndrome as [AIDP] presenting clinically as [GBS]." *Id.* He wrote that his "analysis is affected somewhat by the level of care [Petitioner] was given[,]" arguing that if Petitioner's "symptoms had been recognized as immune-mediated polyneuropathy prior to January 2016," then Petitioner would have received IVIG treatment and her course would have begun to reverse, and therefore timing "would not even be an issue to dispute . . . ." *Id.* at 20.

With his second expert report, Dr. Nadareishvili submitted a paper by Oh et al., in which the authors discuss "subacute inflammatory demyelinating polyneuropathy ("SIDP") and . . . present the diagnostic criteria for this disease." Pet'r's Ex. 39 at 1.[33] The authors coined this term for patients that fell outside of either the GBS or CIDP timing criteria; i.e. "cases [that] had progression of peripheral neuropathy over a [four]-to-[eight] week period . . . ." *Id.* at 4. The authors recommended that the following four criteria be used when making a "definite" SIDP diagnosis:

> The diagnosis of "definite SIDP" was made when all four of the following mandatory criteria were met: 1) progressive motor and/or sensory dysfunction consistent with neuropathy in more than one limb with time to nadir between [four] and [eight] weeks, 2) electrophysiologic evidence of demyelination in at least two nerves, 3) no known etiology of neuropathy other than associated diseases, and 4) no relapse on adequate follow-up.

Id. Dr. Nadareishvili did not assert in any of his expert reports that Petitioner suffered from SIDP.

Dr. Nadareishvili also argued that "the record is clear of infectious causes that would explain [Petitioner's] injury to the exclusion of the vaccination." Pet'r's Ex. 19 at 20. He opined that the vaccination "stands in isolation as the only immune stimulus that would explain an immune-mediated neuropathic process[,]" and that it "was the only antigenic insult for which there is evidence in the medical record throughout September, October, November, or December of 2015." *Id.* While he concedes that there was a "suspicion of a urinary tract infection," Dr. Nadareishvili wrote that "there was no clinical symptoms associated to corroborate an infection, and no objective evidence from ultrasound to support it either." *Id.* He also noted that Petitioner "denied fever, gastrointestinal symptoms, or symptoms of respiratory infection[]" at every appointment during the relevant time period. *Id.* Therefore, he argued that the "vaccination[] stands alone as the only potential causal factor for" Petitioner's GBS.

2. Dr. Chaudry's Expert Reports

Dr. Chaudry wrote that he "agree[s] with Dr. Nadareishvili that [Petitioner] likely suffered from GBS, but [he] disagrees with [Dr. Nadareishvili] that the onset of GBS was within [forty-two] days from the date of vaccination or that [Petitioner's] GBS was caused by the [flu] vaccination." Resp't's Ex. A at 8. He argued that the onset of Petitioner's GBS was "a day or a few days before [January 1, 2016]." *Id.* at 12. He continued, "[t]his would be approximately three months after the [flu] vaccination, which is not an appropriate temporal relationship for [establishing] a causal connection." *Id.* Rather, Dr. Chaudry "agree[s] with [Petitioner's] treating physicians that her intervening symptoms were related to [CTS], severe bilateral knee arthritis, and long standing back issues." *Id.*

In his first expert report, Dr. Chaudry disagreed with many of Dr. Nadareishvili's contentions regarding the timeline of Petitioner's symptom development. As an initial matter, Dr. Chaudry argued that Petitioner was symptomatic prior to receiving the flu vaccination on September 30, 2015. *Id.* at 8. He cited to a visit Petitioner had with Dr. Whitfield on July 23,

[33] S.J. Oh et al., *Subacute Inflammatory Demyelinating Polyneuropathy*, 61 NEUROLOGY 1507 (2003).

2015, where she reported "difficulty going up and down stairs as well as difficulty rising from a seated position[,]" as evidence that Petitioner was symptomatic prior to vaccination. *Id.* (citing Pet'r's Ex. 9 at 12). Dr. Chaudry also noted that Dr. Whitfield "documented severe patellofemoral arthritis of [Petitioner's] bilateral knees[,] which were being treated with injections[]" prior to vaccination. *Id.* In addition, Dr. Chaudry wrote that, on the day of vaccination, Petitioner "not only endorsed a long history of low back pain . . . but also noted that this 'was a little bit more frequent lately.'" *Id.* (quoting Pet'r's Ex. 10 at 42).

Dr. Chaudry argued that Petitioner suffered from CTS after receiving the flu vaccination on September 30, 2015. He submitted an article by Ibrahim et al., which defined CTS as "a compressive neuropathy," or "a mononeuropathy or radiculopathy caused by mechanical distortion produced by a compressive force." Resp't's Ex. C at 1.[34] The authors explained that the "[p]rimary features of CTS include pain in the hand, unpleasant tingling, pain or numbness in the distal distribution of the median nerve (thumb, index, middle finger[,] and the radial side of the ring finger), and a reduction of the grip strength and function of the affected hand." *Id.* at 2. They also noted that CTS "[s]ymptoms tend to be worse at night," and that many CTS patients "describe a phenomenon termed the 'flick sign', in which shaking or flicking their wrists relieves symptoms." *Id.* In terms of treatment, the authors noted that options fall into two categories: "conservative and surgical." *Id.* at 6. They listed "oral and transvenous steroids, corticosteroids, . . . carpal bone mobilisation [sic.] and the use of hand splints[]" as conservative options, and explained that patients using these options tend "to experience[] significant short term benefits," although "their efficacy in the long term remains unclear." *Id.*

The authors of this study also discussed how CTS is diagnosed. *Id.* at 4–6. They wrote that a NCS "is considered to be the gold standard in the diagnosis of CTS[] because it is an objective test that provides information on the physiological health of the median nerve across the carpal tunnel." *Id.* at 5. They explained that, during a NCS, a technician "compar[es] the latency and amplitude of a median nerve segment across the carpal tunnel to another nerve segment that does not go through the carpal tunnel, such as the radial or ulnar nerve." *Id.* Then, the nerve is stimulated by a transcutaneous pulse of electricity, which induces an action potential in the nerve[,]" and "[a] recording electrode . . . detects the wave of depolarization as it passes by the surface electrode." *Id.* They noted that this type of NCS "is the most sensitive and accurate technique, with a sensitivity[35] of [eighty]–[ninety-two percent] and specificity[36] of [eighty]–[ninety-nine percent]." *Id.* The authors also discussed Phalen's and Tinsel's tests, which they described as "[t]he two provocative tests most commonly used in the clinical setting . . . ." *Id.* at 4. They explained that, in a Phalen's test, "the patient is asked to flex their wrist and keep it in that position for [sixty] seconds. A positive response is if it leads to pain or paresthesia in the distribution of the median nerve." *Id.* They noted that the Phalen's test has sensitivity "in the

[34] I. Ibrahin et al., *Carpal Tunnel Syndrome: A Review of the Recent Literature*, 6 (Supp. 1: M8) OPEN ORTHOPEDICS J. 69 (2012).

[35] Sensitivity refers to "the conditional probability that a person having will be correctly identified by a clinical test, i.e., the number of true-positive results divided by the total number with the disease . . . ." *Dorland's* at 1664.

[36] Specificity refers to "the conditional probability that a person not having a disease will be correctly identified by a clinical test, i.e., the number of true-negative results divided by the total number of those without the disease . . . ." *Dorland's* at 1714.

range of [sixty-seven percent–eighty-three percent]," and a specificity that "ranges between [forty percent–ninety-eight percent]." *Id.* The authors further explained that a Tinsel's test "is performed by tapping over the volar surface of the wrist. A positive response [occurs when] this causes paresthesia in the fingers innervated by the median nerve: the thumb, index, middle finger and the radial side of the ring finger." *Id.* They noted that the Tinsel's test "has a sensitivity in the range of [forty-eight percent–seventy-three percent]," and a specificity in the range of "[thirty percent–ninety-four percent]." *Id.*

Dr. Chaudry also submitted an article by R. Shiri in which the author conducted a "systemic review and meta-analysis . . . to assess whether [rheumatoid arthritis ("RA")] and osteoarthritis . . . increase the risk of CTS." Resp't's Ex. O at 2.[37] The author concluded that the "systemic review and meta-analysis found an increased risk of CTS in individuals who suffer from RA or [osteoarthritis]." *Id.* at 7. The author also noted that "it seems that arthritis does predict the development of CTS." *Id.*

In his first expert report, Dr. Chaudry argued that Petitioner's complaints of "numbness in both hands[,] especially the fingertips, and waking up through the night having to shake her hands to get the feeling back[]" made on October 28, 2015, "are indeed classic symptoms of [CTS]." Resp't's Ex. A at 8. He wrote that "[p]ins and needles, tingling, and numbness in distal distribution of the median nerve (thumb, middle[,] and part of [the] ring finger)[,]" as well as "symptoms being worse at night and getting better with shaking are highly typical for CTS." *Id.* He noted that Dr. Whitfield documented that Petitioner had positive Phalen's and Tinsel's tests at a December 1, 2015 appointment. *Id.* (citing Pet'r's Ex. 9 at 6). He also discussed wrist splints, which were unsuccessful in relieving Petitioner's symptoms. *Id.* at 12. He explained that "wrist splints don't always relieve symptoms of [CTS]." *Id.* He also noted that Petitioner "did at some point undergo release of [her CTS] . . ., which may be one reason why her symptoms improved." *Id.*; *see also* Pet'r's Ex. 6 at 328. Dr. Chaudry also drew a connection between Petitioner's arthritis and her CTS, noting that her "arthritis symptoms had become more prominent with severe knee pain, rib pain, [and] back pain, which may be one explanation why her CTS became more symptomatic in October 2015." Resp't's Ex. L at 3.

Dr. Chaudry also took issue with Dr. Nadareishvili's interpretation of Petitioner's nerve conduction/EMG test conducted on December 3, 2015, concluding that Dr. Nadareishvili "reinterprets [the test results] . . . incorrectly." Resp't's Ex. A at 10 (citing Pet'r's Ex. 9 at 37). Dr. Chaudry provided a detailed interpretation of this test. *See* Resp't's Ex. D at 5–7. He wrote that Petitioner's "bilateral ulnar sensory amplitudes are normal . . . whereas [Petitioner's] median sensory amplitude is absent on the right side and reduced on the left." *Id.* at 7. These findings "corresponded with numbness and sensory abnormalities only in the median nerve distribution, [with her] ulnar [nerve] being normal." *Id.* Dr. Chaudry disagreed with Dr. Nadareishvili's interpretation of "minimal reduction (only [one percent]) of amplitudes of right ulnar . . . and left ulnar [nerve] amplitude[s,]" as well as an "[eighty percent] reduction of median amplitudes[,]" as being abnormal. *Id.* Rather, Dr. Chaudry agreed with Dr. Vaught's interpretation that these findings were normal, "given the normal sensory response, normal latency, normal conduction velocity, normal needle EMG of the [first dorsal interosseous] muscle[,] and . . . [Petitioner's] age

[37] R. Shiri, *Arthritis as a Risk Factor for Carpal Tunnel Syndrome: A Meta-Analysis*, 45:5 SCANDINAVIAN J. OF RHEUMATOLOGY 339 (2016).

and history of arthritis in the hands." *Id.* He argued that "[t]he exclusive involvement of bilateral median nerves, with sparing of adjacent ulnar nerves[,] virtually excludes polyneuropathy." *Id.* He noted that two other physicians, Dr. Oar and Dr. Whitfield, both "agreed with the diagnosis of severe [CTS]." *Id.* Dr. Chaudry opined that "GBS cannot be an explanation [for Petitioner's hand symptoms] because of the selective nerve involvement, normal clinical and EMG examination of the rest of the peripheral nervous system, and presentation of weakness over two months after the onset of [Petitioner's] paresthesias." Resp't's Ex. L at 3. Therefore, Dr. Chaudry argued that Petitioner was indeed suffering from CTS after vaccination. Resp't's Ex. A at 10.

While Dr. Chaudry agreed that Petitioner eventually did develop GBS, he argued that the onset of her condition did not occur until much later than under Dr. Nadareishvili's theory. He submitted an article by Vriesendorp in which the author discusses "[t]he clinical features . . . of GBS in adults . . . ." Resp't's Ex. H at 2.[38] The author wrote that "GBS is thought to result from an immune response to a preceding infection that cross-reacts with peripheral nerve components because of molecular mimicry[,]" and that "[a] small percentage of patients develop GBS after another triggering event such as immunization . . . ." *Id.* The author noted that "cardinal clinical features of . . . GBS are progressive, fairly symmetric muscle weakness accompanied by absent or depressed [DTRs]." *Id.* The author also wrote that "GBS is associated with" other clinical features, such as "weakness usually start[ing] in the legs, but it begins in the arms or facial muscles in about [ten] percent of patients[;] . . . [f]acial nerve palsies occur in more than [fifty] percent [of patients; and p]ain due to nerve root inflammation, typically located in the back and extremities, can be a presenting feature and is reported during the acute phase by two-thirds of patients with all forms of GBS." *Id.* at 2–3. The author stated that "GBS usually progress over a period of about two weeks[and b]y four weeks after initial symptoms, [over ninety] percent of GBS patients have reached the nadir of the disease." *Id.* at 3. If the progression of the disease is eight weeks or longer, the author wrote that that disease course "is consistent with [CIDP]." *Id.*

Dr. Chaudry did not believe that Petitioner's development of symptoms post vaccination through the end of 2015 were manifestations of GBS. Resp't's Ex. A at 9–11. He began this discussion by reviewing Petitioner's emergency room visit on November 2, 2015, for back and chest pain. *Id.* at 9. He noted that the records from this visit reflect a "normal" neurological examination. *Id.* He also wrote that doctors noted "[c]hest discomfort for several days and several years of back pain secondary to osteoarthritis and degenerative joint disease . . . ." *Id.* He also noted that Petitioner had a near-syncopal episode during this visit, after which "she noted severe weakness." *Id.* However, Dr. Chaudry argued that "[i]t is not uncommon for patients to feel weak all over after having a near-syncopal episode." *Id.* After another normal neurological evaluation, Petitioner was discharged home. *Id.* Therefore, Dr. Chaudry argued that this weakness was not a sign of GBS because "[p]atients with GBS don't have transient weakness like" what Petitioner experienced. *Id.*

Rather, Dr. Chaudry argued that onset of Petitioner's GBS did not occur until shortly before January 2, 2016, because she "did not lose her ability to ambulate until shortly before she

[38] Francine J. Vriesendorp, *Guillain-Barré Syndrome in Adults: Clinical Features and Diagnosis*, UPTODATE, retrieved from www.uptodate.com/contents/guillain-barre-syndrome-in-adults-clinical-features-and-diagnosis/
print.

presented" on that date. *Id.* at 11. He noted that at each of the ER visits Petitioner had on November 2, 17, and 24, 2015, Petitioner "walked in" and "was documented to have normal examination with normal cranial nerves . . . and no motor or sensory deficit." *Id.* (citing Pet'r's Ex. 4 at 44, 98). In addition, he wrote that Petitioner "was seen on [December 1, 2015,] and did not report any weakness in her upper or lower extremities." *Id.* (citing Petitioner's Ex. 9 at 5). He also noted that a "detailed examination" conducted during a visit on December 22, 2015, "showed normal strength in hip flexion, hip extension, quadriceps, hamstrings, dorsiflexion, and plantar flexion and all reflexes . . . [were] 2+." *Id.* While there was "noted decreased sensation in median nerve distribution[,]" there was "no weakness . . . in [Petitioner's] median, ulnar, and radial muscles . . . ." *Id.* On December 29, 2015, Petitioner "had no cranial nerve deficit and had no motor or sensory deficit[,]" and she was subsequently "discharged home." *Id.* Then, on January 2, 2016, records reflect that Petitioner suffered "acute onset of left[-]sided Bell's [P]alsy at 3 PM [the day prior, as well as] left[-]sided weakness, which was sufficient to warrant consideration of stroke as a possible diagnosis." *Id.* Dr. Chaudry opined that Petitioner's onset date was somewhere in this window.

Dr. Chaudry also reviewed Petitioner's urinary retention issues in his first expert report. *Id.* He disagreed with Dr. Nadareishvili's characterization of this symptom "as being from autonomic dysfunction . . . ." *Id.* Rather, Dr. Chaudry argued that "it is better explained by [Petitioner's] narcotic use given that she had severe constipation as well." *Id.* (citing Pet'r's Ex. 7 at 29).

In addition, Dr. Chaudry also discussed "whether the 'paresthesias about the toes on both of [Petitioner's] feet on December 1, 2015, could be evidence of early onset GBS . . . ." Resp't's Ex. D at 1. Dr. Chaudry described paresthesias as "abnormal sensations experienced in the absence of specific stimuli . . . usually described as a burning, tingling, or numbness." *Id.* He noted that this symptom is "thought to represent abnormal impulses . . . arising from sensory pathways starting from peripheral nerves to the sensory cortex." *Id.* (citing Resp't's Ex. E at 1).[39] When paresthesias occurs because of a peripheral neuropathy, Dr. Chaudry explained that it "should be associated with abnormalities on sensory examination, either reduced pinprick sensation, reduced temperature sensation, reduced vibration sensation, or reduced joint and position[al] sense[,]" as well as with "abnormalities on [DTR] testing[,] especially around the ankles." *Id.* In terms of GBS, Dr. Chaudry wrote that "[p]aresthesias can be part of [the] . . . syndrome[,]" but usually "are persistent, progressive, and occur in the setting of rapidly progressive weakness and areflexia[,]" which "peaks between [two] to [four] weeks" after onset. *Id.* Dr. Chaudry noted that a GBS diagnosis "requires rapidly progressive weakness in the legs and arms and absent reflexes . . . ." *Id.* at 2.

In terms of Petitioner's paresthesias, Dr. Chaudry argued that Petitioner's "one episode of paresthesias in the toes on [December 1, 2015,] was not associated with weakness . . . ." *Id.* In addition, he noted that Petitioner also had a normal examination, including reflexes, on this date. *Id.* Dr. Chaudry opined that, aside from a normal examination and reflexes, "the time frame of evolution of symptoms is inconsistent with [a] GBS diagnosis[]" because "there was no symptom

[39] *Paresthesia Information Page*, National Institute of Neurological Disorders and Stroke (Dec. 29, 2018), retrieved from www.ninds.nih.gov/Disorders/All-Disorders/Paresthesia-Information-Page.

of weakness in the two-month period" between vaccination and the end of December 2015. *Id.* He noted that Petitioner's "weakness was first noted towards the end of December 2015[,] and peaked in early January 2016[,]" which would mean that Petitioner suffered "[t]wo[-]to[-three] months of paresthesias followed by acute onset of weakness . . . ." *Id.* In Dr. Chaudry's opinion, this timeline "is not consistent with a diagnosis of GBS[,] which requires peak of illness to be less than [four] weeks . . . ." *Id.* Dr. Chaudry also listed the following alternative causes for Petitioner's paresthesias: "entrapment neuropathy, lumbosacral radiculopathy, B12 deficiency (high methylmalonic acid . . .), glucose intolerance . . ., anxiety/stress related phenomenon, or central nervous system involvement." *Id.* (citing Pet'r's Ex. 6 at 368; Pet'r's Ex. 10 at 17). Therefore, Dr. Chaudry concluded that the paresthesias noted in Petitioner's December 1, 2015 office visit was not evidence of early onset GBS. *Id.*

Dr. Chaudry concluded his last expert report by writing that, "in [his] professional opinion, [Petitioner] had typical symptoms, signs, and electrophysiology of [CTS] that had symptom onset [on] or about [October 10, 2015]. This CTS had no causal relationship to the flu vaccine. [Petitioner] developed typical symptoms, signs, and electrophysiology of GBS on or about [December 29, 2015]. This GBS occurring over two months after the flu vaccine was not caused by the flu vaccine." Resp't's Ex. L at 4.

IV. Applicable Legal Standards

To receive compensation under the Vaccine Act, Petitioner must demonstrate either that: (1) she suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as amended by 42 C.F.R. § 100.3; or (2) that she suffered an "off-Table injury," one not listed on the Table as a result of her receipt of a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). Petitioner concedes that she cannot meet the Table criteria for flu vaccine-caused GBS. *See* Pet'r's Ex. 19 at 12–13. Thus, she must prove that her GBS was caused-in-fact by the flu vaccine.

To establish causation-in-fact, Petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. 42 U.S.C. § 300aa-13(a)(1)(A). Petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Secretary of the Department of Health and Human Services*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). The *Althen* test requires the petitioner to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. To establish entitlement to compensation under the Program, Petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *See id.*

Under the first *Althen* prong, Petitioner must offer a scientific or medical theory that answers in the affirmative the question "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health and Human Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Rather, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *Id.*

A petitioner is not required to present medical literature or epidemiological studies to meet her burden. *Grant v. Sec'y of Health and Human Servs.*, 956 F.2d 1144, 1149 (Fed. Cir. 1992); *Andreu v. Sec'y Health & Human Servs.*, 569 F.3d 1367, 1380 (Fed. Cir. 2009). However, to the extent medical literature and epidemiological studies are provided, the special master will consider them when deciding whether the petitioner has met her burden of proof. Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu*, 569 F.3d at 1380. This does not negate or reduce a petitioner's ultimate burden to establish her overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

Under *Althen*'s second prong, petitioners must demonstrate that the vaccine actually did cause the alleged injury in a particular case. *See Althen*, 418 F.3d at 1279; *Pafford*, 2004 WL 1717359, at *4. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1380; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain how and why the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original).

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418F.3d at 1280). This is because "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health and Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not "binding on the special

master or court." 42 U.S.C. § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

Under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. For example, if the petitioner's theory involves a process that takes several days to develop after vaccination, an injury that occurred within a day of vaccination would not be temporally consistent with that theory. Conversely, if the theory is one that anticipates a rapid development of a reaction post vaccination, the development of the alleged injury weeks or months post vaccination would not be consistent with that theory. Causation-in-fact cannot be inferred from temporal proximity alone. *See Grant,* 956 F.2d at 1148; *Thibaudeau v. Sec'y of Health & Human Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983) ("Without more, [a] proximate temporal relationship will not support a finding of causation.").

V. Analysis

A. *Althen* Prong One

Petitioner has met her burden with respect to *Althen* prong one. Dr. Nadareishvili presented a detailed, well-reasoned theory based on molecular mimicry whereby the flu vaccine can cause GBS. This occurs when immune system activation caused by the flu vaccine causes a cross-reaction between antibodies to the flu and the peripheral nerves. This cross-reaction results in the antibodies attacking the otherwise healthy cells of the peripheral nerves, resulting in GBS. He provided a detailed discussion of the disease in his expert reports and supported his theory with citations to medical literature, of which multiple were either submitted or cited to by Respondent. This theory is well-established and well-settled in the Vaccine Program. *See*, *e.g.*, *Barone v. Sec'y of Health and Human Servs.*, No. 11-707V, 2014 WL 6834557 (Fed. Cl. Spec. Mstr. Nov. 12, 2014); *Stitt v. Sec'y of Health and Human Servs.*, No. 09-653V, 2013 WL 3356791 (Fed. Cl. Spec. Mstr. May 31, 2013); *Stewart v. Sec'y of Health and Human Servs.*, No. 06-777V, 2011 WL 3241585 (Fed. Cl. Spec. Mstr. July 8, 2011). Respondent did not, either through Dr. Chaudry's expert reports or other filings, present a counter-argument or attempt to discredit Petitioner's general causation theory in any way. Therefore, I find that Petitioner has presented preponderant evidence that the flu vaccine can generally cause GBS.

B. Symptom Onset and Diagnosis

Subsequent to establishing general causation, Petitioner must also establish that her general causation theory is applicable to her symptom onset and diagnoses. Petitioner argues that the first manifestation of her GBS was the hand pain and paresthesias she first mentioned approximately one-month post vaccination. Respondent, however, contends that Petitioner's GBS did not begin until at least December 29, 2015—approximately twelve weeks post vaccination—when she developed weakness in her lower extremities. Respondent further contends that Petitioner's symptoms during the preceding time period were caused by other diagnoses. Therefore, before

determining whether Petitioner has met her burden with respect to *Althen* prong two, I must make a factual determination as to exactly what conditions Petitioner suffered from and when.

While Petitioner argues that her hand pain and paresthesia were not symptoms of CTS, the evidence demonstrates that these symptoms were, more likely than not, manifestations of this condition. The Ibrahim et al. article submitted by Respondent demonstrated that Petitioner's symptoms mirrored the primary features of CTS. Petitioner developed pain and numbness in her hands, especially along the median nerve, which were worse at night and relieved by shaking her hands. These symptoms closely track the description of CTS provided by the Ibrahim et al. study. Additionally, Respondent submitted the Shiri article, which drew a connection between arthritis and CTS. Resp't's Ex. O at 2. Petitioner had severe arthritis for which she received multiple injections during 2014 and 2015. This makes a CTS diagnosis more likely.

Petitioner also met the diagnostic criteria for a CTS diagnosis. She had positive Phalen's and Tinsel's signs at an appointment with Dr. Whitfield on December 1, 2015, which the Ibrahim et al. paper described as the "two provocative tests most commonly used in the clinical setting" to diagnose CTS. Resp't's Ex. C at 4. Dr. Vaught also interpreted Petitioner's EMG/NCS study as being indicative of CTS, because he saw median nerve involvement but no ulnar nerve involvement. In contrast, Petitioner's EMG/NCS study conducted on January 4, 2016, revealed *both* median and ulnar nerve involvement, a marked shift from Petitioner's first EMG/NCS study conducted in December of 2015. A finding of ulnar nerve involvement is not indicative of a CTS diagnosis. In addition to Dr. Vaught, two other treaters diagnosed Petitioner with CTS, namely Dr. Whitfield and Petitioner's PCP. Therefore, based on Petitioner's description of her hand symptoms, the diagnostic evidence, and treaters' diagnoses, I find it more likely than not that Petitioner suffered from CTS in the weeks immediately following her vaccination and prior to her development of symptoms associated with GBS.

Petitioner complained of abdominal and back pain between her vaccination and GBS diagnosis, and she argues these symptoms were manifestations of her GBS. However, Petitioner complained of back pain on the day of vaccination and noted that it was occurring more frequently at that time. *See* Pet'r's Ex. 10 at 42. The timing of this complaint indicates a pre-existing condition. In addition, Petitioner's medical records demonstrate that her treaters diagnosed her abdominal and back pain as either resulting from uncontrolled hypertension or her longstanding osteoarthritis. Petitioner submitted a case report by Wong et al. to support her argument that these symptoms were manifestations of her GBS. The mission of the authors was to "draw attention to abdominal pain as being a presenting symptom[]" of GBS. Pet'r's Ex. 34 at 3. The case report did not discuss statistics regarding how many GBS patients first present with abdominal symptoms nor how many patients develop abdominal pain during their disease course. *See id.* at 1–3. In fact, the authors were unable to say if the case study patient suffered pain due to demyelination or gastrointestinal dysfunction. *Id.* at 2. Therefore, this case report is not enough to establish by a preponderance of the evidence that Petitioner's symptoms of abdominal and back pain were manifestations of her GBS.

Lastly, Petitioner argues that she suffered from autonomic dysfunction, namely uncontrolled hypertension and urinary retention, during her GBS course. Similar to her back pain, one of Petitioner's chief complaints on the day she received the vaccination at issue was hypertension. Pet'r's Ex. 10 at 42. In that visit note, her PCP noted high blood pressure and a history of hypertension since the year 2005. *Id.* Therefore, this condition preexisted the vaccination. In addition, Petitioner only provided a single case report discussing hypertension as a presenting symptom of GBS. *See* Pet'r's Ex. 36. However, Petitioner did not explain how her GBS course is analogous to the case report. Specifically, the patient's symptoms in the case report developed over a period of days, with profound lower extremity weakness developing two days after presentation to the hospital with hypertension and headaches. Petitioner's symptoms developed over a period of months and she provided no context for how the case report is applicable to the facts of her case. As for the symptom of urinary retention, Petitioner did not complain of urinary retention until after she was hospitalized in January of 2016. At her urology appointment on November 30, 2015, Petitioner explicitly *denied* any bladder retention issues. *See* Pet'r's Ex. 5 at 39–40. Petitioner is correct that this visit note lists "flaccid neuropathic bladder" as an active problem; however, Dr. Kowalkowski did not assess Petitioner with this condition nor provide any further context for this notation. Therefore, I find that Petitioner's alleged autonomic dysfunction occurring *before* she was hospitalized in January of 2016 were not manifestations of her GBS.

Based on the foregoing, I find that Petitioner suffered from CTS, back and abdominal pain as a result of osteoarthritis, and urinary issues post vaccination that were not related to her GBS course. With these findings in mind, I will now analyze the evidence as it relates to *Althen* prong two.

C. *Althen* Prong Two

After analyzing the evidence submitted by both parties, I find that Petitioner did not establish by a preponderance of the evidence that her symptoms met the criteria for a GBS diagnosis until January 3, 2016. Dr. Nadareishvili argued in his expert reports that Petitioner suffered from AIDP-GBS. Pet'r's Ex. 19 at 13. Both parties submitted the Willison et al. article, which explained that the two symptoms necessary for a GBS diagnosis in clinical practice are "progressive weakness in the legs and arms" and "areflexia." Pet'r's Ex. 33 at 5; Resp't's Ex. G at 5. Based on the medical records, Petitioner did not develop lower extremity weakness until at least December 29, 2015, when she presented to the emergency room after a fall at church. *See* Pet'r's Ex. 4 at 43. Petitioner was not assessed with lower extremity weakness, however, until December 31, 2015, when she was released from University of Virginia Hospital, Pet'r's Ex. 6 at 463-64, or until January 2, 2016, when she presented to the emergency room with left extremity weakness such that she could not raise her left leg off the hospital bed without support. *See* Pet'r's Ex. 4 at 33. Her treaters did not note areflexia until January 3, 2016, after Petitioner was transferred to St. Mary's Hospital. *See* Pet'r's Ex. 6 at 329. Therefore, based on Petitioner's own medical literature, Petitioner could not have been clinically diagnosed with GBS until at least January 3, 2016.

Petitioner attempts to use her office visits with Dr. Whitfield on December 1 and 22, 2015, as evidence that her GBS began earlier than it was diagnosed. Petitioner's complaints at those visits belie that contention. At her December 1, 2015 office visit, Petitioner did complain of some paresthesias about her toes. *See* Pet'r's Ex. 9 at 5. However, Petitioner explicitly *denied* "any weakness in her upper or lower extremities[]" at this visit. *Id.* Dr. Whitfield did not make any notation in the objective portion of the record that Petitioner had any weakness or areflexia. *See id.* Aside from his assessment of "[l]ower extremity neuropathy of unclear etiology[,]" Dr. Whitefield did not provide any other information about this symptom. *See id.* at 5–9. At her December 22, 2015 office visit, Petitioner did not make any complaints related to her lower extremities. *See id.* at 2. Indeed, Dr. Whitfield did not note any findings of weakness or areflexia in the objective portion of the office visit record. *See id.* at 2–4. In light of Petitioner's complaints at this visit and Dr. Whitfield's notes, it seems that Dr. Whitfield, inadvertently or otherwise, may have copied over his assessment of lower extremity neuropathy from Petitioner's previous visit. There are no notations anywhere else in the record related to Petitioner's lower extremities. *See id.* at 4. Accordingly, I find it more likely than not that Petitioner's GBS did not manifest until December 29, 2015 at the earliest.

In addition to suffering from the conditions described in section V.B. above, Petitioner failed to present persuasive evidence that GBS manifests in a way that is consistent with her symptom development. She argues that the presenting symptom of her GBS was her hand pain and paresthesia; however, this is inconsistent with how a typical GBS case develops based on Petitioner's own filed medical literature. Petitioner's medical literature demonstrated that, in a typical GBS case, the presenting symptom is progressive, ascending bilateral weakness, which usually begins in the distal lower extremities. Respondent also filed medical literature to this effect. Further, Dr. Nadareishvili argued that Petitioner's GBS was a typical AIDP-GBS case. Based on the filed literature and expert reports, Petitioner's symptoms developed in what is an atypical fashion. Dr. Nadareishvili did not present an argument as to why Petitioner's symptoms would develop in such a manner if she suffered from what he described as a typical case. While Dr. Nadareishvili opined that Petitioner's treaters did not diagnose her GBS until January 2016 because they erred in their treatment, s*ee* Pet'r's Ex. 19 at 20 (Dr. Nadareishvili noting that his "analysis [of Petitioner's case] is affected somewhat by the level of care she was given."), this argument is unpersuasive. Dr. Nadareishvili did not support this argument with any citations to medical literature or other supporting documentation.

Petitioner finally attempts to argue that an absence of identified infectious causes in the medical records supports the flu vaccine as the cause of her GBS. However, relying on a temporal relationship and the absence of other known causes is not enough. *See Pafford*, 2014 WL 1717359, at *9 (noting that the "Court has always held that vaccines are not the cause of every event that occurs soon after their administration."). Petitioner has not offered any persuasive scientific evidence that GBS can manifest in the way in which she alleged her symptoms developed. Accordingly, I find that Petitioner has failed to meet her burden under *Althen* prong two.

D. *Althen* Prong Three

As stated, based on the evidence submitted, I find it more likely than not that Petitioner's GBS manifested on approximately December 29, 2015. Accordingly, this is the onset date I will use to analyze Petitioner's claim under *Althen* prong three.

Both parties submitted numerous articles discussing GBS and the timeline for symptom development. The Willison et al. article, which both parties submitted, states that the progressive course of GBS should last days to four weeks. Pet'r's Ex. 33 at 5; Resp't's Ex. G at 5. The authors of that study noted that "[m]ost patients . . . reach the nadir [of weakness] within [two] weeks[,]" although it could "last up to [six] weeks after onset . . . in some rare cases." *Id.* This timeline is consistent throughout the literature. Petitioner began to develop symptoms normally associated as presenting symptoms in GBS approximately three months post vaccination. She provided no evidence that this is an appropriate temporal relationship in vaccine-induced GBS.

While Petitioner argues the appropriate window is longer, the evidence submitted does not support her position. Petitioner did submit evidence that SIDP could develop in an extended timeframe; however, Dr. Nadareishvili explicitly argued that Petitioner developed AIDP-GBS, not SIDP. Therefore, per Petitioner's own expert, this article is inapplicable to Petitioner's case. No other piece of medical literature submitted by either party supports a twelve-week symptom development of GBS. In addition, while not binding on my analysis, no other decision in the Vaccine Program has found that GBS onset occurring twelve-weeks post flu vaccination is appropriate for entitlement to compensation. *See Barone*, 2014 WL 6834557, at *13 (stating that eight weeks is the longest reasonable timeframe for a flu vaccine-induced GBS and citing cases). Therefore, I find that she has not met her burden under *Althen* prong three.

VI. Conclusion

For the foregoing reasons, Petitioner's claim is hereby DISMISSED for insufficient proof. The Clerk of Court is directed to enter judgment accordingly.[40]

IT IS SO ORDERED.

s/Herbrina D. Sanders
Herbrina D. Sanders
Special Master

[40] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.